IMPACT OF TMI NUCLEAR ACCIDENT UPON PREGNANCY OUTCOME, CONGENITAL HYPOTHYROIDISM AND INFANT MORTALITY*

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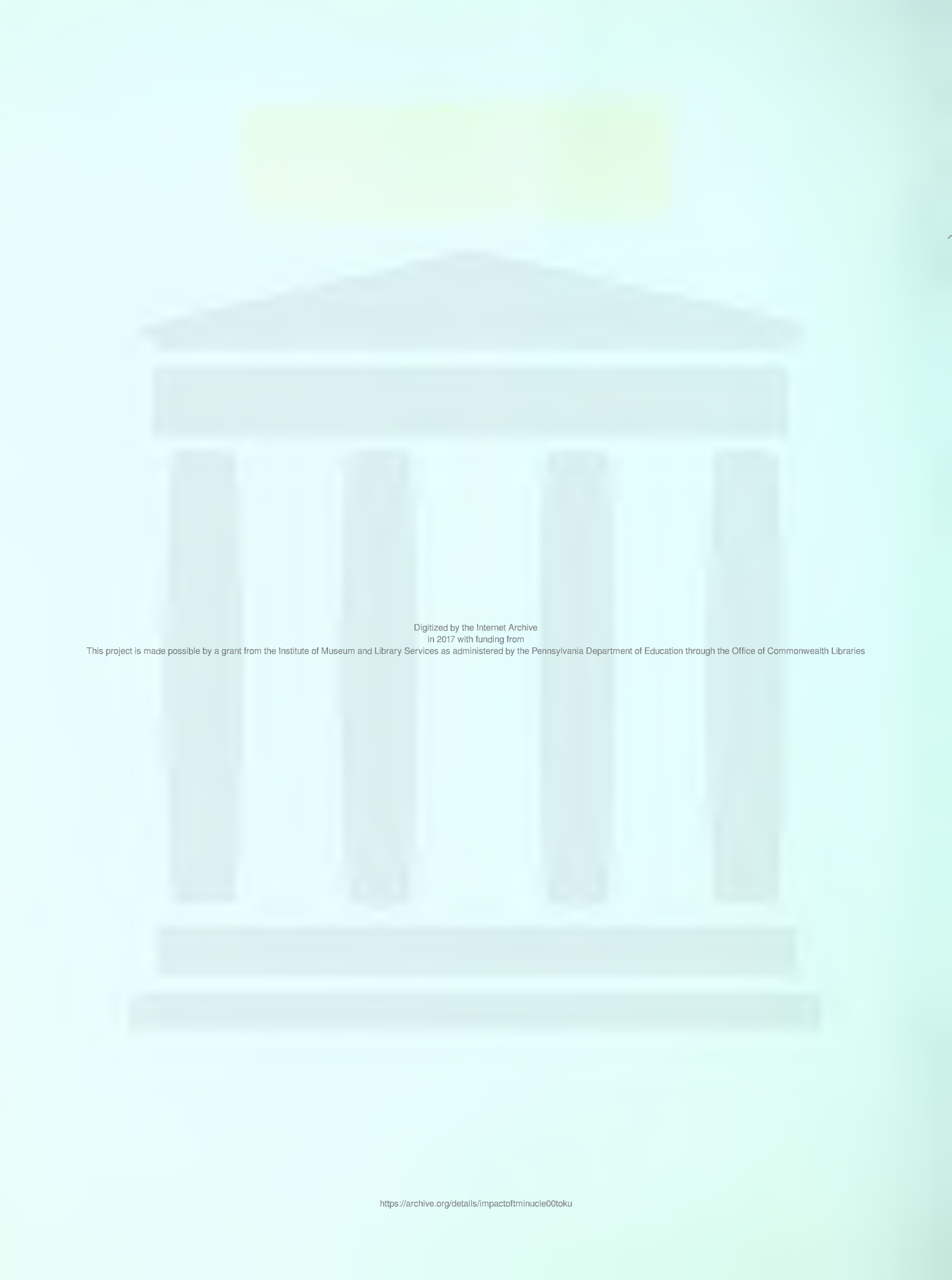

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The Three Mile Island nuclear accident of March 28, 1979 has resulted in marked social unrest locally, nationally and world-wide, particularly with respect to the health and safety aspects of nuclear energy. Subsequent to the accident the Pennsylvania Department of Health initiated a comprehensive evaluation of possible health effects of the accident upon local population. During the 10-day period of crisis, it was not possible to ascertain accurate information regarding radioactive emissions from the damaged nuclear reactor into the environment. However, the presence of rather diffuse and growing psychological disturbance in the area was apparent.

Within a short period of days following the accident, we were able to conceptualize and develop a multidisciplinary plan for a variety of different research studies specifically designed to assess the health impact of the TMI accident. Studies conceived during this critical period mostly reflected the existing epidemiological knowledge regarding biological effects of low level ionizing radiation and severe emotional stress.

I was designated by the Governor of Pennsylvania to coordinate and manage all health-related research activities relative to TMI. At the same time, a special Advisory Panel was commissioned by the Secretary of Health to oversee and guide all TMI-related studies administered by the Department of Health.

A. PREGNANCY OUTCOME AROUND THREE MILE ISLAND

One of the most important studies developed shortly after the accident was to determine if the TMI nuclear accident has had any measurable impacts upon pregnancy outcome and infant health in the vicinity of the damaged nuclear reactor. We knew that both ionizing radiation and emotional stress can affect human reproductive process and pregnancy outcome. We also recognized that the embryo and the fetus are highly sensitive to such environmental insults, depending upon their severity, the mode of exposure, and the gestational age when exposed.

Before describing the methodology and study design in detail, let me review briefly the current state of epidemiology of pregnancy outcome, particularly in relation to radiation and stress.

Radiation and Pregnancy Outcome:

Much information is now available regarding the effects of ionizing radiation on the embryo and fetus. Most of the more reliable data are derived from animal experiment; however, certain experimental findings may be applicable to the humans, at least in a qualitative sense, while recognizing inherent limitations or difficulties in such cross-species inferences.

The most significant damage from exposure to ionizing radiation results from the direct interaction of the stream of ions produced by radiation with the nucleus of the irradiated cells. The cell may be killed, the radiation may produce no damage, or such damage may be repaired. There is another type of damage which is probably the most significant one, i.e., the damaged cell survives and reproduces a clone of abnormal cells which may result in malignancies or congenital anomalies.

Possible effects of radiation on pregnancy outcome are (a) intrauterine and extrauterine growth retardation, (b) embryonic, fetal or neonatal death, and (c) gross congenital malformations. The tissue (organ) most readily and consistently affected by radiation is the *central nervous system.*

Laboratory and clinical studies by and large support the contention that doses of radiation less than 10 rads do not contribute to intrauterine or extrauterine growth retardation or to gross congenital malformations(1). Distribution of the absorbed dose from X-rays or gamma rays externally exposed is considered to be rather uniform in the developing embryo or fetus; thus, a child with multiple radiation-induced malformations is also likely to have intrauterine growth retardation and some CNS abnormalities.

To determine the effect of radiation upon pregnancy outcome, one must consider (a) the absorbed dose, (b) the dose rate (acute or chronic; continuous or intermittent), (c) the stage of gestation at which the exposure occurred, (d) the age of the mother when conceived, and (e) the health condition of the mother, in general. If the dose rate is reduced significantly, the damaged cell may recover from it in time. The pre-implanted stage of the embryo is the most sensitive to lethal effects of radiation. Embryos destroyed at this stage of pregnancy may never be recognized or recorded. However, preplantation irradiation has no apparent relationship to teratogenesis. Radiation has its greatest effectiveness in producing *congenital malformations* during the organogenesis period. In humans, this corresponds to the 14th-49th day of gestation(2).

The peak incidence of *gross malformations* occurs when the fetus is irradiated during the early organogenesis period, although cellular, tissue and organ *hypoplasia*, including *growth retardation* can be produced by radiation throughout organogenesis, and fetal and neonatal periods, if the dose is high enough. These are usually limited to CNS abnormalities and other organs, which continue to differentiate throughout gestation. Thus, *cerebral hypoplasia*, *microcephaly*, *cerebellar hypoplasia*, and *testicular atrophy* can be produced by "high" doses at specific stages of gestation.

A number of studies suggest that "low" levels of radiation with less than 10 rads of acute or chronic exposure may produce some pathologic effects in the embryo(3), (4), (5), (6), but these minor effects may be subtle and thus difficult to detect. For this reason, the National Council on Radiation Protection and Measurements has established the *maximum permissible dose* (MPD) to the fetus from occupational exposure of the expectant mother well below the known teratogenic dose. The *neonatal death* rate is highest in the surviving embryos irradiated during the early organogenesis period.

Radioactive isotopes administered internally to the pregnant woman have a variable distribution in the embryo and fetus depending upon (a) the stage of gestation, (b) whether the radioactive material crosses the placenta, and (c) the biochemical affinities of the type of radiation emitted (alpha, beta, or gamma). Thus, the evaluation of the relative risk is much more complex and difficult for radiations absorbed from internally administered radioactive materials than for radiation delivered from external X-rays or gamma ray sources.

It is generally assumed that *embryonic germ cells* are susceptible to the mutagenic effects of radiation throughout gestation. However, there is some uncertainty as to whether "low" doses below 10 R or low dose rates can produce significant cytogenetic defects. There has been no convincing evidence that cytogenetic (chromosome) abnormalities as such caused by radiation in utero have caused any significant increase in the incidence of clinical diseases.

There is no doubt that "high" doses of radiation can be carcinogenic. However, whether or not "low" doses, such as below 2 rads, can induce leukemia and/or other malignant tumors in the humans has been debated by some epidemiologists and radiation biologists. It appears improbable that radioactive fallout as reported in the past or natural background radiation as such, significantly affects the incidence of *congenital malformations*, *growth retardation* or *fetal death*.

The exact nature and extent of damages caused by "low" doses of radiation upon humans are still unknown. However, if the cell nucleus is damaged by radiation and some genetic materials (DNA) are lost or impaired, one may not conclude that the risk is zero. It is logical to assume then, that there is no threshold in radiation effect which may increase more or less with the increase or accumulation of exposure. However, the problem we are facing today is that anomalies caused by such "low" doses of radiation, if any, may not be detectable with the existing method of epidemiologic inquiry.

It is also important to recognize that not all persons run the same risk of developing a malignancy or other abnormalities from a given radiation exposure. These variations depend upon individual genetic-constitutional makeups, as well as different individual experiences and environmental exposures.

Stress and Pregnancy Outcome:

Stress or psychoemotional disturbance is considered by many researchers as a precursor to disease. There are a number of studies in humans which have found an association between prenatal anxiety/stress and gestational, perinatal and developmental pathology. While some of these studies seem to have methodological flaws, several have found a significant relationship to either complications of pregnancy(7), (8), or to infant growth and development(9).

Nuckolls, in particular, studied the effect of "social support" upon pregnancy outcome(10). Women with a high number of "psychological assets" had one third the pregnancy and perinatal complication rate of women whose "psychological assets" were low.

Newton(11) in a retrospective study of postpartum women showed that pregnancies terminating in premature labor were more likely to have been stressful. In terms of the average number of life events per pregnancy, it was clear that the more premature the onset of labor, the higher the level of psychological stress was likely to be. The groups were matched for age, gravidity, and parity. The results of the study were independent of socioeconomic levels.

The findings of the studies cited above suggest a number of practical and scientific questions to be addressed within the context of TMI Health Effect Research Program. The *first*, and most obvious question, is whether or not the local population, including pregnant women, *as a whole* experienced any detectable stress effects. Previous studies of stress and pregnancy complications have found relationships which are either relatively weak(12) or restricted to subgroup of the overall study population.

A *second* question concerns factors which render individual women, particularly vulnerable to stress effects. As reviewed earlier, stress may be associated with morbidity only in the absence of supportive interpersonal relations. This observation is in accord with other studies of stress and illness(13), as well as with Burchfield's(14) argument that a maladaptive response to stress is atypical, and likely to occur only when adequate coping resources are unavailable.

An assessment of the role of social support, as well as other possible mediating factors, would contribute to the study of stress as a scientific concept and provide information as to which segments of the pregnant population might be at risk for stress-induced morbidity.

While specific mechanism of stress-induced morbidity is not yet fully understood, there may be several different explanations with respect to pregnancy outcome; e.g., stress-anxiety induced changes (a) in maternal behavior, such as increased smoking, drinking or medication during pregnancy, (b) in obstetric practice, such as increased prescription of analgesics and psychotropic drugs or use of special procedures, (c) in maternal-infant bonding and child-rearing practices, and (d) in the hypothalamic-adrenocortical mechanisms(15).

A carefully designed retrospective cohort study of Pregnancy Outcome was initiated in August, 1979 following four months of preparation. This study covered all pregnant women residing within a 20-mile radius of the TMI, who gave births during a one-year period from March 28, 1979 through March 27, 1980. This study cohort consisting of approximately 4,000 deliveries will be compared with a control cohort of another 4,000 deliveries during a one-year period immediately following the study cohort in the same geographic area. The study cohort will also be compared with similar data collected in the same general area during the immediately preceding four-year period. This design will make it possible to compare pregnancy outcome measures among three cohorts, one study group and before-and-after control groups.

Measures of adverse pregnancy outcome being investigated are: fetal deaths (stillbirths with and without abortions of 16-week or more gestation) as expressed per 1,000 deliveries, neonatal deaths (deaths within 28 days postpartum) as expressed per 1,000 live births, hebdomadal deaths (deaths within seven days postpartum) as expressed per 1,000 live births, perinatal deaths (combined measure of fetal and neonatal deaths) as expressed per 1,000 deliveries, prematurity (gestation less than 37 weeks) as expressed in percent, immaturity (birth weight less than 2,500 grams) as expressed in percent, congenital malformations (one or more defects observed at birth) as expressed in percent, and low Apgar score (less than seven at one minute of delivery) as expressed in percent.

As indicated earlier, the main objective of the present investigation is to determine if the TMI nuclear accident has had a measurable effect on pregnancy outcome. However, there are numerous factors other than radiation and stress that are known or suspected to influence the course of pregnancy and fetal outcome. In order to delineate the effect of the TMI accident, other known influences must be taken into account. This necessitated ascertainment of the appropriate data on a large number of variables pertaining to the pregnant women themselves and the surrounding complex environment to which they have been exposed.

The *maternal factors* considered in this study include: *sociodemographic* characteristics, such as race, age, education, occupation, employment, marital status, religion and residence; *behavioral attributes* such as smoking, drinking, and birth control practice; and *medical-obstetric histories*, such as diabetes, hypertension, thyroid disease, obesity, previous abortions-miscarriages, previous fetal deaths, prematurity/immaturity, congenital malformations, and gravidity/index birth order.

The *provider factors* that were taken into account are: *medical specialty* of the attending physician (obstetrician; general-family practitioner; osteopath; etc.); *type of practice* (solo vs. group); and *prenatal care* (initiation of medical care, frequency of visits, special procedures or tests done, instructions given, medications administered, X-ray exposures, etc.).

Maternal stress during the index pregnancy is being measured by overt personal statements of "anxiety-fear" as experienced by individual women during the crisis, as well as by actual stress-coping patterns, such as taking tranquilizers and sleeping pills. *Maternal radiation exposure* during the 10-day crisis following the nuclear accident is being estimated by the Department of Radiation Health of the University of Pittsburgh Graduate School of Public Health. For this purpose, all available and reliable radiation source data compiled by various agencies are being reviewed carefully, consolidated and computer analyzed on the digitized electronic maps with respect to distance and direction of each pregnant woman from the Three Mile Island. Also, added to this body of data is detailed data relative to individual whereabouts during the 10-day period so that more accurate radiation exposure can be

estimated. Eventually, two series of dose estimates on an individual basis will be established, namely, *maximum possible dose* and *most likely dose.*

These *radiation dose estimates*, together with the measures of psycho*logical stress* will be related to each of the eight pregnancy outcome measures, while holding constant influences of all other factors considered in the study. The impact of the TMI nuclear accident will be assessed in terms of both radiation and stress combined, as well as each factor considered independently.

Since the level of radiation exposure is considered to be very low and thus no major radiation effect upon pregnancy outcome is expected. On the other hand, it is possible that some measurable radiation effects might be detected if the originally "reported" radiation dose data were significantly underestimated. Our ability to detect relatively small differences, if any, in the incidence of adverse consequences attributable to the nuclear accident will heavily depend upon how well we can control (take into account) the influences of all the other factors, some of which are known to be much more important than low level radiation and/or psychological stress being investigated.

B. CONGENITAL HYPOTHYROIDISM

One of the important radioactive releases during the TMI nuclear accident was that of I^{131}. Since it is known that *radioactive iodine* can cause hypothyroidism and that I^{131} can be taken up by pregnant women in the vicinity of TMI which, in turn, absorbed by the fetal thyroid gland through placenta after the 10th week of gestation, we decided to examine the incidence of *congenital hypothyroidism* among newborn infants. The fetal thyroid gland is much more sensitive to radioactive iodine than is the mother's thyroid gland (fetal thyroid affinity for iodine is greater than maternal thyroid affinity); i.e., a relatively small dose to the mother can be a relatively large dose to the fetus.

Beginning in July 1978, all children born alive in Pennsylvania are required to be screened for hypothyroidism, the condition characterized by lack of or insufficient level of thyroid hormone in the infant's blood. The purpose of this screening program is to find newborns with metabolic defects early enough after birth to prevent mental retardation.

The thyroid screening procedure involves testing for "low" thyroxine (T4) and "high" pituitary thyroid stimulating hormone(TSH). Confirmation of diagnosis is done through thyroid scan, which can help determine various types of the abnormality. During the initial six-month period, testing procedures were not fully standardized and the results were not considered to be complete.

There are several different diagnostic classes in congenital hypothyroidism: (a) *agenesis* (absence of the thyroid gland), (b) *dysgenesis or ectopic type* (incomplete maturation and/or displacement of the thyroid gland from the normal position), (c) *dyshormonogenesis or genetic type* (lack of enzyme necessary to synthesize thyroxine and/or difficulty in the release mechanism of thyroxine; the condition usually inherited from the parents as an autosomal recessive trait) and (d) *other types* (abnormalities caused by environmental agents). In a normal population, the incidence of congenital-neonatal hypothyroidism is in a range of one in 4,500 to 5,000 live births.

During the March 28, 1979-March 27, 1980 period only one case of congenital hypothyroidism was identified within a ten-mile radius of TMI among approximately 4,000 newborn infants. This incidence rate is well within a normal range of expectation.

The Statewide incidence of congenital hypothyroidism for 1979 (12-month period) was one per 4,600 live births, which is also within a normal range of expectation. The rate for 1978 (only the latter six-month period) was considerably lower; this was expected because of the fact that the thyroid screening program in Pennsylvania was started in July 1978 and that during this start-up period the screening procedures and standards were not yet fully established, making data unsuitable for comparison. The Statewide incidence for 1980 was one per 4,427 live births, again indicating that the level of congenital hypothyroidism for Pennsylvania as a whole remained within a normal range.

An apparent clustering of seven cases of congenital hypothyroidism in Lancaster County during 1979 was subjected to a special in-depth analysis and investigation because of physical proximity and timing of the Three Mile Island nuclear accident. The following diagnostic and epidemiological features are of interest: (a) One of the seven cases identified was reported in January of 1979, prior to the TMI accident, thus has no connection with radioactive iodine released from the damaged nuclear reactor. (b) One with severe multiple central nervous system anomalies was born three months after the accident; this case is unlikely to be associated with TMI accident because of the late gestation period of the fetus when the nuclear accident occurred (most, if not all, of these defects would have come about prior to the TMI accident) and also of coexisting developmental anomalies which are unlikely to be associated with radiation. (c) One case was of dysgenesis, representing one of discordant Amish twins, thus, non-supportive of the etiology secondary to radiation exposure. (d) Another case of dysgenesis in whom the thyroid glands were displaced from the normal position. (e) One case of dyshormonogenesis from an Amish family where the condition (lack of enzyme to synthesize thyroxine) was inherited from the parents. (f) For the remaining two cases no thyroid scan was conducted.

Having completed detailed diagnostic analysis and epidemiological assessment of all the cases reported in Lancaster during 1979, we concluded that cases of congenital hypothyroidism were not related to the TMI nuclear accident. Except for the two cases for which diagnostic scan was not performed (unknown type), these types of anomalies are not expected to result from direct or indirect exposure of the fetus to radioiodine. This conclusion was also supported by an independent Hypothyroidism Investigative Committee organized by the State Health Department, which included expertise in the fields of epidemiology, pediatric endocrinology, obstetrics, medical genetics, biostatistics, and radiation physics.

Apart from the incidence analysis presented above, there is also an important consideration with respect to radiation in relation to congenital hypothyroidism.

First, after March 28 through December 31, 1979, no single case of congenital hypthyroidism was reported in Dauphin, Cumberland, Perry, Northumberland, Juniata, Snyder, Mifflin, and Union Counties,

the areas downwind (N, NW, NNW) from the Three Mile Island during the first 48 hours of the accident, when probably the largest amount of radioactive releases took place, thus the largest amount of contamination including I^{131}.

Second, the maximum combined (inhalation and ingestion) *human thyroid dose* of radioactive iodine in the vicinity of the TMI following the March 28, 1979 accident through April 1979 is estimated to be 7.5 mrad (Editorial: **Annals** of Internal **Medicine**, Vol. 91, No. 3, September 1979). At least 1,000 times greater thyroid doses (i.e., 7.5 rads) would be required to have significant acute damages to the thyroid gland; however, even at this dose level, many of the damaged cells may be repaired. Based on the experiences of the Marshallese exposed to fresh radioactive fallout and atomic bomb victims, it is considered likely that as much as 50 to 100 rads fetal thyroid doses would be necessary to cause irreversible tissue damages, such as congenital hypothyroidism and/or thyroid cancer. Acknowledging the fact that the *fetal thyroid* is much more sensitive to radio-iodine than is the *maternal thyroid* (a conservative upper bound estimate is that the thyroid dose to a fetus may be as high as ten times the maternal thyroid dose), the maximum likely *fetal thyroid dose* (approximately 75 mrad) and the maximum possible thyroid dose of 190 to 200 mrad in the vicinity of the damaged nuclear plant are still far too small to have caused congenital hypothyroidism.

In an epidemiological investigation of possible "clustering" of a disease or morbid condition, it is important to recognize the technical difficulty and methodological limitations associated with such investigation. It is the overall consistent pattern of observation that provides useful basis for conclusion, rather than a single isolated change or difference, which in most cases occurs without substantive epidemiologic significance. This is particularly true when relatively small populations are being studied. One may or may not find a "statistically significant" change, difference, or clustering in morbid rates in an area depending upon how such population is delineated geographically and/or temporally. It is equally important that investigators carefully examine the observed relationships and determine if such relationships are consistent with the known biological theory or orientation, which is based on the previous studies and experiences. Our conclusions regarding congenital hypothyroidism around the Three Mile Island nuclear plant have been based on both the overall pattern of epidemiologic observations and in reference to the existing scientific knowledge.

C. INFANT MORTALITY

Ionizing radiation is often related to infant morbidity and mortality in a general context of biological effects because of the greater sensitivity of the newborns to radiation, as compared with the adult population. The infant mortality is defined as the risk of infants dying within the first year of life and is expressed per 1,000 live births.

Subsequent to the March 1979 nuclear accident, we initiated a comprehensive evaluation of the existing vital statistics data in order to determine if the TMI accident has had any measurable influence upon infant mortality in the vicinity of the damaged plant.

For the purpose of the present study, we considered a 10-mile radius of the Three Mile Island, wherein approximately 4,000 infants are born annually (Table 1). Both levels of radiation exposure and psychological distress within the 10-mile radius communities were higher than those beyond the 10-mile radius communities. The available mortality data were analyzed by calendar quarters, as well as annually, for each of the three consecutive years, 1977, 1978, and 1979, for the entire 10-mile area, including Harrisburg, the 10-mile area excluding Harrisburg, and Harrisburg separately. For cross-sectional comparison, corresponding mortality data for the State of Pennsylvania as a whole, were evaluated for the same historical time frames.

As indicated in Table 2, the *infant mortality rate* was not significantly different between the 10-mile area with or without Harrisburg and the State of Pennsylvania for any of the three years under consideration. The higher infant death rate indicated for Harrisburg separately is a reflection of the fact that approximately one-half of the infants born in the city were nonwhite.

The infant mortality rate within the 10-mile radius, including Harrisburg, was already considerably high (19.3 per 1,000 live births) during the *first quarter* of 1979 prior to the TMI accident. The rate remained at the same level during the *second quarter* of 1979 immediately following the accident, but declined substantially during the *third* 12.7) and *fourth* (13.4) *quarters.* This temporal pattern of change in the rate is consistent with the view that the TMI accident has had no measurable impact upon infant mortality. Otherwise, the infant mortality rate would have increased steadily (or, at least, would have remained high as a result of interaction between seasonal downward trend and TMI-related upward trend), particularly during the third and early fourth quarters. Fetal sensitivity to radiation and maternal distress is much greater in the earlier period of gestation or organogenesis when exposed and this would have been reflected on the gradually rising mortality trend following the accident for a period of nine to ten months. However, the actual observation was contrary to this hypothesis.

Within the 10-mile radius of TMI, the 1979 infant mortality rate (16.1) was not significantly different from the 1977 rate (12.5). The 1978 infant mortality rate (10.8) in the same area was somewhat atypical and unusually low, particularly within the immediately surrounding communities outside of Harrisburg (8.4). This is largely because of the small population, wherein marked statistical variations from year to year are not at all uncommon with no particular epidemiologic significance. For this reason, the 1978 infant mortality rate should not be used as a normal base for comparison.

Having considered both cross-sectional and temporal analyses of the available vital statistics data compiled by the State Health Department, we found no evidence that the TMI nuclear accident has had any significant impact upon infant mortality. Statistical variations or differences, as observed in the 10-mile radius, are considered to be a typical random phenomenon in a relatively small population with no particular epidemiologic significance. Theoretically, too, the low levels of radiation exposure, as reported offsite, cannot be directly related to such massive destruction or impairment of cells that cause infant deaths.

The pattern of *fetal mortality rate* or the risk of the fetus being born dead in the vicinity of the Three Mile Island was also analyzed by the same method as applied to infant mortality. We found that there is no indication that the TMI nuclear accident was related to its quarterly or annual variations in fetal mortality (Table 3). The level of fetal mortality within the 10-mile communities was, in fact, considerably lower than that for the State as a whole.

SUMMARY AND CONCLUSIONS

The Three Mile Island nuclear accident has caused an extensive social and political unrest world-wide. At the same time, it has presented social scientists and biomedical investigators a unique opportunity to evaluate its impact upon local population. Probably the most important concern is that of safety and health effects of this unprecedented event.

From the currently available epidemiological knowledge, no significant physical health effects are expected from the low level radiation reported to have been released from the damaged TMI facility. However, in the absence of absolute certainty as to the exact amount of radioactive contamination of the local environment and population, particularly during the early period following the accident, carefully designed epidemiological studies, such as those described in this report, are justified. Furthermore, some substantial psychological impacts upon local populace have been documented. It is not known at this time what significant physical manifestations, if any, may actually ensue from psychological distress over an extended period of years. In addition, because of high sensitivity of the fetus to ionizing radiation and severe maternal stress, timely evaluation of pregnancy outcome should be pursued.

Based on the already established TMI Population Registry, there should be a continuous and long-term epidemiologic surveillance of the exposed general population, which includes approximately 37,000 individuals. The TMI Population Registry is now updated annually so that annual mortality (rate and cause) and periodic health survey, can be conducted. Such endeavor will make it possible to determine and document if there is any measurable health impact in humans from the low level of ionizing radiation that has not yet been fully studied.

It is the responsibility of government agencies and academic communities to properly inform the general public with the objective results of carefully-designed scientific studies of possible health effects of the TMI nuclear accident. It is also critical that safety of nuclear energy is properly addressed in relation to that of other available means of energy production, as well as many other potential risks in human life. Equally important is the public understanding of various alternatives, so that the society as a whole, rather than selected few, can make a rational choice for its constituents. It is hoped that TMI Health Effect Research Program will serve as a means to achieve such goals.

TABLE 1: RESIDENT LIVE BIRTHS BY QUARTER: PENNSYLVANIA AND TEN MILE TMI AREA COMMUNITIES, 1977-1979

Year/Quarter	Pennsylvania	TMI Ten Mile Area Total	Harrisburg City	Excluding Hbg. City
1977	**(153,415)**	**(3,750)**	**(1,001)**	**(2,749)**
Jan. – March	36,911	886	242	644
April– June	38,414	937	248	689
July – Sept.	40,181	977	274	703
Oct. – Dec.	37,909	950	237	713
1978	**(151,438)**	**(3,803)**	**(1,057)**	**(2,746)**
Jan. – March	37,084	926	261	665
April– June	36,339	922	262	660
July – Sept.	39,932	1,029	302	727
Oct. – Dec.	38,083	926	232	694
1979	**(157,533)**	**(3,905)**	**(1,185)**	**(2,720)**
Jan. – March	38,326	932	296	636
April– June	38,351	983	303	680
July – Sept.	41,933	1,023	302	721
Oct. – Dec.	38,923	967	284	683

TABLE 2: RESIDENT INFANT DEATHS, NUMBER AND RATE, BY QUARTER: PENNSYLVANIA AND TEN MILE TMI AREA COMMUNITIES, 1977-1979

Year/Quarter	Number of Deaths				Death Rate Per 1,000 Live Births			
		Ten Mile TMI Area				Ten Mile TMI Area		
	Pa.	Total	Harrisburg City	Excluding Hbg. City	Pa.	Total	Harrisburg City	Excluding Hbg. City
				Infant Deaths				
1977	**(2,137)**	**(47)**	**(15)**	**(32)**	**(13.9)**	**(12.5)**	**(15.0)**	**(11.6)**
Jan. — March	544	13	6	7	14.7	14.7	24.8	10.9
April— June	554	11	2	9	14.4	11.7	8.1	13.1
July — Sept.	520	9	3	6	12.9	9.2	10.9	8.5
Oct. — Dec.	519	14	4	10	13.7	14.7	16.9	14.0
1978	**(2,031)**	**(41)**	**(18)**	**(23)**	**(13.4)**	**(10.8)**	**(17.0)**	**(8.4)**
Jan. — March	530	13	8	5	14.3	14.0	30.7	7.5
April— June	509	9	3	6	14.0	9.8	11.5	9.1
July — Sept.	473	5	1	4	11.8	4.9	3.3	5.5
Oct. — Dec.	519	14	6	8	13.6	15.1	25.9	11.5
1979	**(2,118)***	**(63)**	**(31)**	**(32**	**(13.4)**	**(16.1)**	**(26.2)**	**(11.8)**
Jan. — March	511	18	10	8	13.3	19.3	33.8	12.6
April— June	537	19	9	10	14.0	19.3	29.7	14.7
July — Sept.	507	13	3	10	12.1	12.7	9.9	13.9
Oct. — Dec.	562	13	9	4	14.4	13.4	31.7	5.9

* *Includes one death, month of occurrence, unknown.*

TABLE 3: RESIDENT FETAL DEATHS (TOTAL), NUMBER AND RATE, BY QUARTER: PENNSYLVANIA AND TEN MILE TMI AREA COMMUNITIES, 1977-1979

Year/Quarter	Number of Deaths				Death Rate Per 1,000 Deliveries*			
		Ten Mile TMI Area				Ten Mile TMI Area		
	Pa.	Total	Harrisburg City	Excluding Hbg. City	Pa.	Total	Harrisburg City	Excluding Hbg. City
	Fetal Deaths (Total)							
1977	**(4,058)**	**(80)**	**(45)**	**(35)**	**(25.8)**	**(20.9)**	**(43.0)**	**(12.6)**
Jan. — March	1,062	16	10	6	28.0	17.7	39.7	9.2
April— June	992	18	9	9	25.2	18.8	35.0	12.9
July — Sept.	1,026	23	12	11	24.9	23.0	49.0	15.4
Oct. — Dec.	978	23	14	9	25.1	23.6	55.8	12.5
1978	**(4,034)**	**(77)**	**(38)**	**(39)**	**(25.9)**	**(19.8)**	**(34.7)**	**(14.0)**
Jan. — March	1,003	15	5	10	26.3	15.9	18.8	14.8
April— June	1,047	21	12	9	28.0	22.3	43.8	13.5
July — Sept.	1,001	20	10	10	24.5	19.1	32.1	13.6
Oct. — Dec.	983	21	11	10	25.2	22.2	45.3	14.2
1979	**(3,608)**	**(67)**	**(37)**	**(30)**	**(22.4)**	**(16.9)**	**(30.3)**	**(10.9)**
Jan. — March	938	24	13	11	23.9	25.1	42.1	17.0
April— June	916	12	6	6	23.3	12.1	19.4	8.7
July — Sept.	937	16	10	6	21.9	15.4	32.1	8.3
Oct. — Dec.	817	15	8	7	20.6	15.3	27.4	10.1

* *Deliveries: Live births and fetal deaths (including abortions).*

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